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A multivariate observational analysis on the relationship between coffee consumption and cigarette smoking to blood lipids, hemodynamics, cardiac rate, and respiratory functions

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A MULTIVARIATE OBSERVATIONAL ANALYSIS ON THE RELATIONSHIP
BETWEEN COFFEE CONSUMPTION AND CIGARETTE SMOKING TO
BLOOD LIPIDS, HEMODYNAMICS, CARDIAC RATE, AND RESPIRATORY
FUNCTIONS

Iowa State University

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A multivariate observational analysis on the relationship
between coffee consumption and cigarette smoking to blood lipids,
hemodynamics, cardiac rate, and respiratory functions

by

David Arnold Ludwig

A Dissertation Submitted to the
Graduate Faculty in Partial Fulfillment of the
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INTRODUCTION

Numerous epidemiological studies have examined the relationship between the occurrence of coronary heart disease, coffee consumption, and cigarette smoking. These studies utilize observational data collected on large groups of people who for one reason or another are available for evaluation. Most of the research concerned with the long term effects of coffee consumption have concentrated on caffeine as a risk factor in the incidence of ischemic heart disease. Fewer studies are available which examine the effects of coffee consumption on more precise factors such as blood pressure, blood lipids, or respiratory function. The effects of cigarette smoking have been more widely examined, especially in the area of respiratory illness. Since smoking and coffee drinking have gained the reputation of "social evils", the amount of research on their effects has remained relatively constant over the past 20 years.

Coffee consumption

During the 1976 Health Interview Survey a representative cross-sectional sample of approximately 113,000 noninstitutionalized people living in the United States were asked questions about the use of cigarettes, coffee, aspirin, and sleeping pills (Bonham & Leaverton, 1979). Results indicated that coffee was drunk by 80 percent of the U.S. population 20 years of age and over, with coffee drinkers consuming an average of 3.2 cups each day. Persons who smoked were also likely to drink large amounts of coffee. The study concluded that there is no evidence that heavy coffee drinking, five or more cups per

day, is related to poor health. This was concluded after a comparison of coffee consumption to health related indicators such as chronic conditions, restricted activity days, bed days, and hospitalization days.

After reviewing both experimental and epidemiological research along with a thorough review of the pharmacologic literature, MacCornack (1977) concluded that the ingestion of coffee and cola beverages under "normal" circumstances does not produce permanent detrimental effects. Similar comments on the reappraisal and exoneration of coffee as a risk factor for myocardial infarction and sudden death are expressed in an editorial by Vaisrub (1975b). Regan (1979) states that when used in moderate amounts, coffee does not precipitate cardiac arrhythmias in healthy persons who have no underlying cardiac risk factors. It seems that caffeine containing beverages are innocuous when used in the absence of other risk factors. However, data on the effects of inordinate consumption of coffee have not been available for study.

Prospective analysis of a prevalence survey conducted in Evans County, Georgia, between 1967 and 1969 showed no association with coffee usage in four race sex groups when the effects of cigarette smoking were controlled (Heyden, Tyroler, Heiss, Hames & Bartel, 1978). Neither coronary heart disease nor cerebrovascular death rates were related to coffee drinking habits. Similar findings have been reported by Yano, Rhoads, and Kagan (1977), Hennekens, Drolette, Jesse, Davies, and Hutchison (1976), and Dawber, Kannel, and Gordon (1974) using samples from Hawaii, Florida, and Massachusetts, respectively.

Research concerned with specific physiological changes brought on by the ingestion of coffee or caffeine are sparse. Most of these studies provide information as to acute effects and have minimal applications from an epidemiological standpoint. Robertson, Trolick, Carr, Watson, Hollifield, Shand and Oates (1978) have shown that a 250 mg dosage of caffeine produces a mean elevation in blood pressure of 14/10 mm Hg 60 minutes after ingestion. Subsequently, blood pressure gradually moderates over a 3 hour period. It was concluded that borderline hypertensive individuals may benefit by abstaining from caffeine containing beverages. This conclusion was quickly attacked by Bertrand, Pamper, Hillman, Duffy and Michell (1978) on the grounds that long term studies have failed to demonstrate any relationship between daily coffee consumption and blood pressure elevation. Bertrand et al. (1978) also present data on 72,101 employees of the IBM Corporation which illustrate a lack of association between blood pressure and coffee consumption.

The current opinion on the relationship between coffee consumption and coronary heart disease held by the majority of medical professionals can be best summarized in a quote by Vaisrub (1975a).

At a time when our life's pleasurable habits are undergoing a process of attrition, when many culinary delights are proscribed, it is comforting to know that a cup of coffee, sine sugar and cream of course, need not be a medical "no-no".

Cigarette smoking

Research concerned with the effects of cigarette smoking on cardio-pulmonary functions is much more voluminous and diverse. Respiratory disturbances are by far the most widely examined ramifications of cigarette smoking. However, other physiological changes such as blood lipids and blood pressure have also been intensively studied.

Smoking and forced expiratory volume at one second (FEV_1)

One of the simplest measures of respiratory function is FEV_1 . This test relates the volume of gas expelled by an individual under maximal exhalation over one second of time. Usually expressed as a percentage, FEV_1 is the ratio of actual volume of gas expelled to expected volume of gas expelled, as predicted from nomographs using information about the subject's age and height.

McCarthy, Craig, and Cherniack (1976) studied the effect of smoking cessation on FEV_1 in 49 men and 82 women. Fifty-nine of the subjects stopped smoking for intervals lasting between six and 24 weeks. Only a slight increase in FEV_1 was noted as the length of cessation increased. Similar results were reported earlier in the Framingham cohort where it was concluded that obstructive disease caused by cigarette smoking is usually limited to peripheral airways, a condition not readily detectable by air FEV_1 (Ashley, Kannel, Sorlie & Masson, 1975). Peripheral airways are less than two millimeters in diameter and contribute in only a small way to airway resistance. This makes possible a significant progression of disease without a change in FEV_1 , which reflects overall airway resistance. These findings led

Brooks, Zipp, Barber, and Carson (1978) to test FEV_1 in cigarette smokers and nonsmokers using gases of varying density. No significant difference was found between smokers and nonsmokers for expiratory flows while breathing air. Forced expirations using higher density gases such as helium proved to be much more sensitive in discriminating between smokers and nonsmokers. This differentiation was due to the diffusion properties of higher density gases into the peripheral airways. Air was too dense and could only reflect large airway obstruction not typically caused by smoking cigarettes.

An interesting result has been replicated in studies which examine the relationship between smoking habits, age and FEV_1 . Hamby (1975) found a pronounced but statistically nonsignificant interaction between age and three smoking classifications. The ability of FEV_1 to differentiate smokers, nonsmokers, and exsmokers was poor at younger ages but improved for ages over 50. This finding was also seen in a study by Bosse, Costa, Cohen, and Podolsky (1975). Tockman, Menkes, Cohen, Permutt, Benjamin, Ball, and Tonascia (1976) concluded, based on similar findings, that FEV_1 probably reflects the effects of continued smoking and demonstrates increasing abnormalities associated with longer exposure.

Plasma lipids Because of their role in the atherosclerotic process, the serum lipids have been among the most widely investigated risk factor thought to be associated with the development of coronary heart disease (Castelli, Doyle, Gordon, Hames, Hjortland, Hulley, Kagan

& Zukel, 1977; Kannel, Castelli & Gordon, 1979). Attempts to find factors associated with blood lipid levels have been conducted in the hope that subsequent relationships will provide further information regarding the etiology of atherosclerosis. Many variables have been studied, with the relationship between cigarette smoking and serum lipids currently attracting a great deal of interest.

Hjermann, Helgeland, Holme, Lund-Larsen, and Leren (1976) demonstrated a slight positive correlation ($r = .05$), between total serum cholesterol and increasing daily exposure to cigarette smoke. It was suggested, however, that elevated cholesterol may be a result of possible dietary differences, since food preference may differ according to smoking habits. More recent studies have focused on the relationship between cigarette smoking and high density serum lipids (HDL-c), since this lipid fraction appears to be a protective factor against coronary heart disease. HDL-c was studied in 4107 middle-aged men and women in relation to cigarette usage in the Framingham Offspring Study (Garrison, Kannel, Feinleib, Castelli, McNamara & Padgett, 1978). A negative association was found between HDL-c and cigarette smoking after adjusting for alcohol consumption. This result was comparable across both sexes, with average differences ranging from 4 mg/dl in men to 6 mg/dl in women. Results also indicated that this effect may be reversible since former smokers had about the same HDL-c levels as nonsmokers. Heyden, Heiss, Manegold, Tyroler, Hames, Bartel, and Cooper (1979) also observed a decrease in HDL-c with increased cigarette usage. This trend, however, was not statistically

significant and the authors refrained from speculating on the causal physiological mechanism due to the observational nature of the investigation. Decreases in HDL-c levels also seemed to be accompanied by an increase in triglyceride levels, though differences were again statistically nonsignificant. Similar results were reported by Morrison, Kelly, Mellies, deGroot, Khoury, Gartside, and Glueck (1979). In a study of 965 school children, adolescents who smoked had significantly lower levels of HDL-c and higher levels of low density lipoproteins than nonsmokers.

Confounding factors that also seem to influence plasma HDL-c levels are consumption of alcohol and exercise (Castelli et al, 1977; Wood & Haskell, 1979). Both of these activities are associated with increasing plasma HDL-c levels. It is plausible that smoking is related to a sedentary life style and an increased tendency to consume alcoholic beverages.

Overall, the high density lipoprotein fraction seems to differentiate smokers and nonsmokers. Low density lipoprotein, total cholesterol, and triglyceride are to a lesser extent, if at all, affected by smoking cigarettes.

Blood pressure Large sample epidemiological studies which have attempted to detect long term effects of smoking cigarettes on blood pressure are of great interest since high levels of systolic and diastolic pressure are believed to be deleterious. Although the acute effects of smoking cigarettes are well-known, the long term effects are still being considered.

Gyntelberg and Meyer (1974) examined the relationship between blood pressure and smoking after accounting for physical fitness, relative weight, and alcohol consumption. The results obtained from 5249 middle aged males indicated a decrease in both systolic and diastolic pressure among subjects who smoked. Differences between smoker's and nonsmoker's systolic and diastolic pressure were approximately 4 mm Hg and 3 mm Hg, respectively. These results were replicated by Seltzer (1974) in a study of 849 adult white male veterans who participated in an ongoing prospective study on aging. Additional analysis indicated that subjects who managed to quit smoking experienced a sharp rise in both systolic and diastolic pressures. Why these changes occur are not fully known, although an adaptive condition similar to responses after exercise have been postulated (Gyntelberg & Meyer, 1974).

Response to exercise and resting heart rate A precise quantitative definition of physical fitness is difficult, due to the many physiological parameters thought to be associated with an individual's ability to handle increasingly greater work loads. Physiological measurements taken during maximal exertion are considered to be among the most valid indicators of aerobic power and are commonly used to reflect possible cardiorespiratory changes brought about by smoking cigarettes. Since the heart adapts to aerobic training, cardiac rate during rest is also used as an indicator of physical fitness.

In a cross-sectional study of 1093 men of various occupational backgrounds, Blackburn, Brozek, and Taylor (1960) studied the relation-

ship between smoking intensity, resting heart rate, and circulatory reactions to submaximal exercise. Resting heart rate was found to be only slightly higher in smokers than nonsmokers. Differences in various circulatory responses including heart rate during exercise were also found to be minimal. In most instances, statistical differences were detected only when heavy smokers were compared to subjects who never smoked. The authors concluded that there were no large or important differences between smokers' and nonsmokers' performing exercise tests. Chevalier, Bowers, Bondurant, and Ross (1963) found no statistical differences in maximal oxygen uptake ($\dot{V}O_2$ max) between 18 smokers and 14 nonsmokers, although $\dot{V}O_2$ max values were slightly higher in nonsmokers. Most of the differences between groups could be attributed to a significantly greater accumulation of oxygen debt by smokers during exercise. Heart rate during rest and exercise was found to be significantly higher in smokers by approximately 10 beats per minute. Krumholz, Chevalier, and Ross (1964) also found that smokers and nonsmokers had comparable $\dot{V}O_2$ max values with oxygen debt being significantly higher in smokers. No differences, however, were seen in heart rate during rest or exercise. In a companion study, 10 subjects were evaluated before and after abstinence from cigarette smoking (Krumholz, Chevalier, & Ross, 1965). Again, significant changes were seen in oxygen debt but not $\dot{V}O_2$ max. Lower heart rates after cessation of smoking were observed during rest and the last minute of a five minute bicycle ergometer test. Exercise heart rates at three minutes did not differ. Frayser (1974) studied the response of 10 smokers and

seven nonsmokers, all of which were females, to a month of repetitive exercise. Pretraining evaluation indicated no differences in $\dot{V}O_2$ max between the two groups. Oxygen debt, however, was significantly lower among smokers. Kerrigan, Jain, and Doyle (1968) found that control heart rates and systolic blood pressures of 25 smokers and 10 nonsmokers did not differ prior to, during, or after exercise. In an observational study of 586 male members of the Indiana State Police Force, McHenry, Faris, Jordan, and Morris (1977) found that smokers had slightly higher systolic blood pressures and slightly lower heart rates than nonsmokers during maximal exercise. Additional findings indicated that the prevalence of exercise induced ventricular premature complexes was not influenced by smoking habits.

The literature tends to indicate that smokers and nonsmokers do not differ, at least to any significant degree, in their response to maximal or submaximal exercise. There is some indication, however, that smokers experience a higher degree of distress during recovery.

Epidemiological research, although useful, can only provide hints as to the causal effects drinking coffee and smoking cigarettes may have on the cardiorespiratory system. Since researchers involved in observational studies cannot manipulate and control variables as one does in laboratory experiments, final conclusions can only be achieved through numerous replications. The problem of concomitant factors, which may influence the results, can to some extent be controlled by various multivariate statistical methods.

It is, therefore, the purpose of this study to investigate the effects of coffee consumption and cigarette smoking on blood lipids, hemodynamics, cardiac rate, and respiratory function. These effects will be evaluated after individual variation due to age, weight, activity level, and body composition is removed.

Hypotheses

In accordance with the previous literature, the following research hypotheses are advanced:

1. No overall relationship will be found between the number of cups of coffee drunk per day and any of the nine dependent variables under study. Dependent effects include; (a) FEV₁, (b) total serum cholesterol, (c) triglycerides, (d) HDL-c, (e) resting systolic blood pressure, (f) resting diastolic blood pressure, (g) resting heart rate, (h) $\dot{V}O_2$ max, and (i) double product (max. systolic pressure x max. heart rate).
2. Smokers will have lower levels of HDL-c than nonsmokers. This difference is hypothesized to be between 3 and 5 mg/dl.
3. Total serum cholesterol and triglycerides will not differ in smokers and nonsmokers.
4. Smokers and nonsmokers will not differ in their FEV₁ percentages.
5. Systolic and diastolic blood pressures are hypothesized to differ by approximately 4 mm Hg and 3 mm Hg, respectively, with smokers having lower values than nonsmokers.

6. Since past research on resting heart rate has for the most part has been inconclusive, it is hypothesized that the resting heart rates of smokers and nonsmokers will not differ.

7. No differences in $\dot{V}O_2$ max will exist between smokers and non-smokers.

In view of the lack of clear-cut relevant research, differences in double product and the possible effect of coffee consumption by cigarette smoking interactions will be regarded as exploratory. Interactions involving selected covariates will also be examined in a secondary analysis to insure the validity of the statistical procedures.

METHODS

Subjects

Initially 555 male employees of the Iowa Department of Public Safety were available for study. The majority of the subjects were highway patrolmen with the balance being made up of fire marshalls, capital security employees, and officers of the Department of Criminal Investigation. Subjects were participants in a 52 week prescribed exercise training program conducted by the Iowa State University Exercise Clinic. Since multivariate statistics were used, only cases with complete data could be analyzed. This left a total of 478 subjects for the study. Ages were reported as of the last birthday and ranged from 19 years to 64 years with a mean of 38.0 years and a standard deviation of 9.55 years.

Procedures and materials

Informed consent and general information questionnaire All subjects read and signed an Informed Consent Form before any data were collected (see Appendix A). A general information questionnaire was completed by all participants (see Appendix B). Information pertinent to coffee consumption, cigarette smoking, and daily activity level was obtained from this questionnaire. Subjects indicated how many cups of coffee they were currently consuming per week. Subjects were categorized into one of the following usage groups: (a) non-drinkers; zero cups per week, (b) light drinkers; one to 14 cups per week, (c) average drinkers; 15 to 28 cups per week, (d) above average drinkers; 29 to 42 cups per week, and (e) heavy drinkers; more than 42 cups per week. Though categories of usage vary from study to study,

the categories chosen for this study are an attempted consolidation of classification schemes used in previous research (Dawber et al., 1974; Hennekens et al., 1976; Jick, Miettinen, Neff, Shapiro, Heinonen & Slone, 1973). Subjects were classified as smokers if they reported to be currently smoking cigarettes. Otherwise, subjects were classified as nonsmokers. A dichotomy was chosen for smoking status to avoid the small cell frequencies which resulted when attempting to differentiate light and heavy smokers. Subjects were considered active if they reported regularly running or walking at least three miles per week or were currently involved in an organized exercise program. They were considered sedentary if neither of these activities was indicated. Although activity level is probably measured with some degree of error, with some of the initial bias remaining after adjustment, it was felt that this bias would not be large enough to make tests of significance unreliable.

Anthropometric measurements Weight was measured to the nearest kilogram by a Health-O-Meter medical scale (model 230KG). A Harpenden skin fold caliper was used to measure skin fold thicknesses. Caliper readings were converted to percent body fat by a formula developed by Pollock et al. (1976). Three determinations were taken at each skin fold site and then averaged to increase the validity of the procedure. To increase the reliability of the skin fold measurements, the same individual took all readings on all participants.

Forced expiratory volume at one second FEV₁ was measured with a six liter Collins wet seal spirometer (model P5150). Maximal effort after three trials was expressed as a percentage of predicted performance normalized for differences due to age and height (Kory et al. 1961).

Resting systolic and diastolic blood pressure Measurement of resting systolic and diastolic blood pressure was performed by an

Iowa State University physician during a limited physical examination conducted prior to the graded exercise stress test. All resting blood pressures were taken with a Taylor Instruments Tykos sphygmomanometer and a Littman Instruments anesthesiologist's stethoscope while subjects were in a standing position.

Stress test and electrocardiogram The graded exercise stress test was conducted in accordance with the modified Sheffield Bruce protocol (Sheffield & Roitman, 1976). Subjects were informed not to participate in any energetic activity or ingest any food or liquid, except water, for a period of 12 hours preceding the graded exercise stress test. It was also requested that the subjects not smoke prior to being tested. All stress tests were administered between the hours of 8:00 a.m. and 11:30 a.m. at the Iowa State University Student Health Services Building. Temperature within the Health Services Building was kept at 68⁰F. Two large fans were used to help circulate the air in the stress testing area. Heart rate was monitored continuously at rest, during exercise, and during recovery by an International Medical Corporation Viagraph (model 120). Resting heart rate was taken while subjects were lying quietly in a supine position. Ten Viatrode disposable electrodes (model 8008-B) were placed on the chest as described by Marriott (1972, pp. 1-8). Electrode sites were prepared in the usual manner by scrubbing with soap and water, shaving and abrading the skin, and cleansing with alcohol. All subjects were taken as close as possible to their predicted maximal heart rate using treadmill procedures developed by Bruce, Kusumi, and Hosmer (1973). An

International Medical Corporation motor driven treadmill (model 200) was used for all graded exercise stress tests. Maximal oxygen uptake was predicted from the subject's performance on the treadmill test (Bruce et al., 1973). Systolic blood pressure was taken every three minutes during the entire procedure.

Plasma lipids Fasting blood samples were drawn from the cephalic vein while subjects were in a semibasal state. Sampling was done by a licensed medical technologist at Iowa State University Student Health Center. In an effort to eliminate variability due to diurnal effects, blood samples were taken at the same time of day. Samples were analyzed on the same day as bleeding by Clinical Laboratories, Ames, Iowa. Variability in the lipid determinations was made available by Clinical Laboratories for purposes of assessing the quality control of the procedures (see Appendix C). Total serum cholesterol was determined by enzymatic hydrolysis of cholesterol esters and enzymatic oxidation of free cholesterol to yield a colored quinaneimine dye (Leon & Stasiw, 1976). Serum triglycerides were determined by an enzymatic method which uses lipase, glycerol kinase, pyruvate kinase, and lactate dehydrogenase to yield lactate and nicotinamide adenine dinucleotide (NAD). The amount of converted NAD is measured spectrophotometrically at 340 nm and is inversely proportional to the amount of triglycerides present in the serum (Bucoło & David, 1973). High density lipoprotein cholesterol was determined by Mg-dextran SO_4 precipitation and enzymatic assay for cholesterol (Finley, Schiffman, Williams & Lichti, 1978).

Variable re-expression

The frequency distributions of the nine dependent variables and three of the four covariables were examined for departures from normality. Cochran (1947) has indicated that the additional time spent on the re-expression of skewed distributions is well worth the effort. Improvement in variable distributions tends to produce more efficient estimates and helps eliminate problems arising during the later stages of data analysis. A square root transformation tended to improve the mild positive skew of total serum cholesterol. A natural log transformation of triglycerides, HDL-c, systolic blood pressure, diastolic blood pressure and resting heart rate was used to remove a positive skewness in the raw measures. Since body fat and FEV₁ were expressed as percentages, angular (arc sine) and logit transformations were examined. Percent body fat was best re-expressed through an angular transformation where the logit seemed to be more appropriate for the re-expression of FEV₁. Double product, $\dot{V}O_2$ max, age, and weight were not transformed since their distributions were normal. Table 1 defines and summarizes the distributions of all variables used in this study.

Statistical analysis

Analysis of the data was broken down into three parts: (a) preliminary analysis of the zero order intercorrelations between the independent variables, covariates, and dependent effects, (b) primary analysis of the effects of coffee consumption and cigarette smoking along with their interaction, and (c) follow-up analysis of residuals and possible covariate interactions with the independent effects.

Table 1
Variable Description and Summarization

Variable name	<u>Description of distribution</u>	
	Original units of measurement	Transformation
Covariates		
Weight	kilograms	none
Age	years	none
Percent body fat	percentage	angular
Activity level	dichotomy; f active = 136, 28.45% f inactive = 342, 71.55%	
Independent variables		
Coffee usage	five categories; f nondrinkers = 73, 15.27% f light drinkers = 102, 21.34% f average drinkers = 132, 27.62% f above average drinkers = 108, 22.59% f heavy drinkers = 63, 13.18%	
Smoking status	dichotomy; f smokers = 215, 44.98% f nonsmokers = 263, 55.02%	
Dependent variables		
Total serum cholesterol	mg/dl	square root
Triglycerides	mg/dl	natural log

Table 1 (continued)

Variable name	<u>Description of distribution</u>	
	Original units of measurement	Transformation
High density lipoprotein, (HDL-c)	mg/dl	natural log
Forced expiratory volume at one second, (FEV ₁)	percentage	logit
Double product, maximal heart rate X maximal systolic blood pressure ÷ 100	$\frac{(\text{beats/min}) \times (\text{mm Hg})}{100}$	none
Systolic blood pressure	mm Hg	natural log
Diastolic blood pressure	mm Hg	natural log
Maximal oxygen uptake, ($\dot{V}O_2$ max)	liters/min	none
Resting heart rate	beats/min	natural log

Preliminary analysis consists of testing the dependent variables zero order correlations. Bartlett's chi-squared statistic (1954) was used to test the hypothesis that the population correlation matrix was equal to identity. Morrison (1976, p. 116) points out that if dependent variable population correlation coefficients are equal to zero, multivariate analysis based upon the sample covariance matrices would be in vain or even misleading. The consequences of performing a multivariate analysis under the condition of zero population correlations are discussed in greater detail by Healy (1969) and Rao (1966, pp. 87-103). Zero order correlations between independent effects and covariates were examined to investigate the possibility of multicollinearity. Cohen and Cohen (1975, pp. 84-91) have demonstrated how erroneous results can be obtained when independent effects are highly correlated.

Primary analysis utilized multivariate analysis of covariance (MANCOVA). The Hotelling-Lawley trace statistic was used to test overall multivariate effects. In the event of a significant MANCOVA, univariate tests are utilized for follow-up analysis. All effects tested were evaluated after all other effects in the model were removed (partial solution). The technique of using stepwise discriminant function as a follow-up to a significant MANCOVA was also utilized (Borgen & Seling, 1978). Unlike the univariate ANOVAs which are limited to specifying the independent contribution of each variable to group separation, discriminant analysis is capable of showing the underlying dimensionality (redundancy) of the data.

In the follow-up analysis, residuals were analyzed to evaluate the assumptions of normality of errors and equality of variance. Two factor interactions between covariates and independent variables were examined to assure the validity of the covariance analysis.

A type I error rate of .05 was used in evaluating all test statistics. This level was chosen after considering the number of subjects available for analysis, the cost of making an error of the first or second type, and the observational nature of the data. Two sided alternative hypotheses were used for all tests since previous research has indicated some conflicting results. These two tailed tests also provide a more conservative approach for declaring statistically significant effects.

Statistical power

Based on 2200 observations, Rosenman, Sholtz, and Brand (1976) have estimated standard deviations for systolic and diastolic blood pressure to be 14.2 and 9.6 mm Hg, respectively. Likewise, studies done by Wood and Haskell (1979) and Morrison et al. (1979) have estimated the standard deviation of HDL to be about 12 ml/dl. Given the hypothesized differences thought to exist between smokers and non-smokers, standardized effect sizes for systolic blood pressure, diastolic blood pressure, and HDL are .33, .28, and .31, respectively. Cohen (1977, pp. 24-27) has indicated that a value of .30 is generally regarded as a small to medium effect. That is approximately 20 percent of the combined area of the smokers and nonsmokers distributions are nonoverlapping. If a 5 percent reduction in explained variance can

be attributed to the covariates, then the power associated with the univariate analysis of covariance is approximately .93. This is based on a sample size in each group of 237 which is the harmonic mean of the number of smokers and nonsmokers available for this study. Since the ratio of nonsmokers to smokers was close to one (1.2), the reduction in power due to the unequal class frequencies is at most .01.

The power of the MANCOVA was not as easily calculated as in the univariate situation. Since the power function of T^2 depends not only on the variances but the correlation structure of the dependent effects, the population variance-covariance matrix was required. Due to the large number of dependent effects and the lack of previous research involving some of the variables, construction of this matrix would involve little more than a guess. Morrison (1976, pp. 160-164) has shown, however, that when the univariate power is high, the sample size is large (>60), and the correlation between effects thought to differ is greater than zero, the power of the multivariate test is comparable to that of the univariate.

RESULTS

Preliminary analysis

The correlations among independent and dependent variables are presented in Tables 2 and 3, respectively. Correlation coefficients involving coffee usage categories are squared coefficients of determinations from the one way analysis of variance after coffee usage (independent effect) was dummy coded. The signs for these coefficients were determined from other discrete measures of association such as Gamma and Kendall's Tau-B. Smokers and active subjects were coded as one, while nonsmokers and inactive subjects were coded as zero. All other coefficients, aside from those obtained from the analysis of variance, are Pearson Product-moment correlations.

The correlation structure of the independent variables seems to indicate that smokers drink more coffee and tend to be less active than nonsmokers, whereas coffee drinkers are older and tend to be heavier with a higher degree of body fat than nondrinkers. These relationships are consistent with findings from the National Health Survey (Bonham & Leaverton, 1979), indicating similar anthropometric and behavioral patterns despite the nonrandom selection process of the present investigation. Multicollinearity would not seem to be a problem since the absolute values of all correlation coefficients are less than .45.

Associations between dependent variables reflect well-documented theoretical relationships. Moderate to high correlations exist within blood lipid fractions, fitness measures (double product and $\dot{V}O_2$

Table 2
Correlations Between Independent Variables

	Age	AngBF	Activity level	Coffee	Smoking
Weight	.219	.434	-.046	.103 ^a	.038
Age		.443	-.076	.304 ^a	.033
AngBF			-.159	.149 ^a	-.001
Activity level				-.098 ^a	-.104
Coffee					.308 ^a

Note. Critical value for $p < .05$ is $\pm .09$ for Pearson Product-moment correlations ($df = 476$).

^aCorrelation coefficients were calculated from the square root of the coefficient of determination in the one-way ANOVA. Multiple correlations involving weight and activity level are statistically nonsignificant ($p > .05$).

Table 3
Correlations Between Dependent Variables

	LgTrig	LgHDL	LgtFEV ₁	DbProd	LgSysBP	LgDiaBP	$\dot{V}O_{2\max}$	LgRstHR
SRChol	.404	.018	-.013	.084	.087	.199	-.236	.152
LgTrig		-.405	-.048	.116	.195	.239	-.244	.125
LgHDL			-.063	.007	-.027	.024	.081	.037
LgtFEV ₁				.069	-.023	-.005	.126	-.031
DbProd					.264	.158	.311	.074
LgSysBP						.711	-.206	.106
LgDiaBP							-.276	.127
$\dot{V}O_{2\max}$								-.222

Note. Critical value for $p < .05$ is $\pm .09$ (df = 476)

max), and blood pressures. Bartlett's chi-square statistic indicates that the null hypothesis of zero correlations between dependent variables is not tenable, $\chi^2(36) = 822.56$, $p < .0001$. Thus, inferences made from multivariate statistics should not be misleading.

Primary analysis

The results of the MANCOVA are summarized in Table 4. The null hypothesis of no overall coffee usage by smoking status interaction seems to be tenable as does the null hypothesis of no overall main effect of coffee usage. Since the probability associated with the test statistic for smoking status is less than .05, the null hypothesis of a common mean vector for smokers and nonsmokers is rejected. Multi- and univariate tests of the main effects were conducted after the interaction term was removed from the model since the main effects are correlated with the interaction. Cohen (1978) has warned that the simultaneous analysis of independent effects and their corresponding interactions generally results in the distortion of the main effects. Adjusted means by smoking status and coffee usage are presented in graphic form for all nine dependent variables in Figures 1 through 9. Figure 1 also provides information as to the number of subjects contributing to each smoking status by coffee usage mean. Re-expressed variables have been converted back to their original units of measurement for ease of interpretation. Follow-up univariate analysis revealed differences in HDL, systolic blood pressure, diastolic blood pressure, and $\dot{V}O_2$ max between smokers and nonsmokers. Adjusted main effect means and standard errors for smoking status along with the

Table 4
 Multivariate Analysis of Covariance for the Overall
 Effects of Coffee Usage, Smoking Status, and Coffee Usage
 by Smoking Status Interaction

Coffee usage

Hotelling Lawley trace = .0758
 \underline{F} approximation with 36 and 1834 \underline{df} = 0.96
 Probability of a greater \underline{F} = .5291

Smoking status

Hotelling Lawley trace = .0741
 \underline{F} approximation with 9 and 460 \underline{df} = 3.79
 Probability of a greater \underline{F} = .0001

Coffee usage x smoking status

Hotelling Lawley trace = .0913
 \underline{F} approximation with 36 and 1818 \underline{df} = 1.15
 Probability of a greater \underline{F} = .2459

Note. Since a partial solution was used, the two factor interaction was removed from the model before evaluating the main effects of coffee usage and smoking status. This was done in order to avoid having the interaction preceding the main effects.

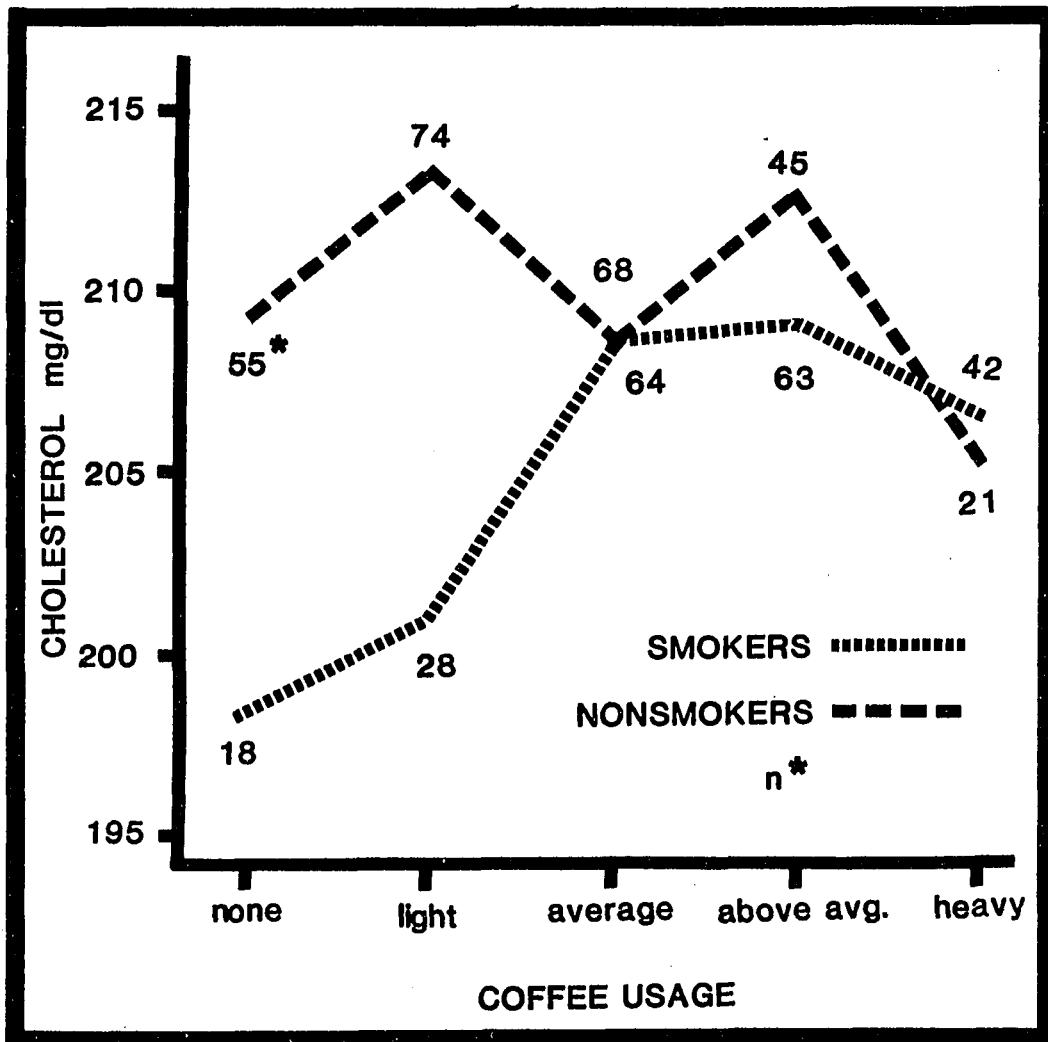


Figure 1. Cholesterol means by coffee usage and smoking status

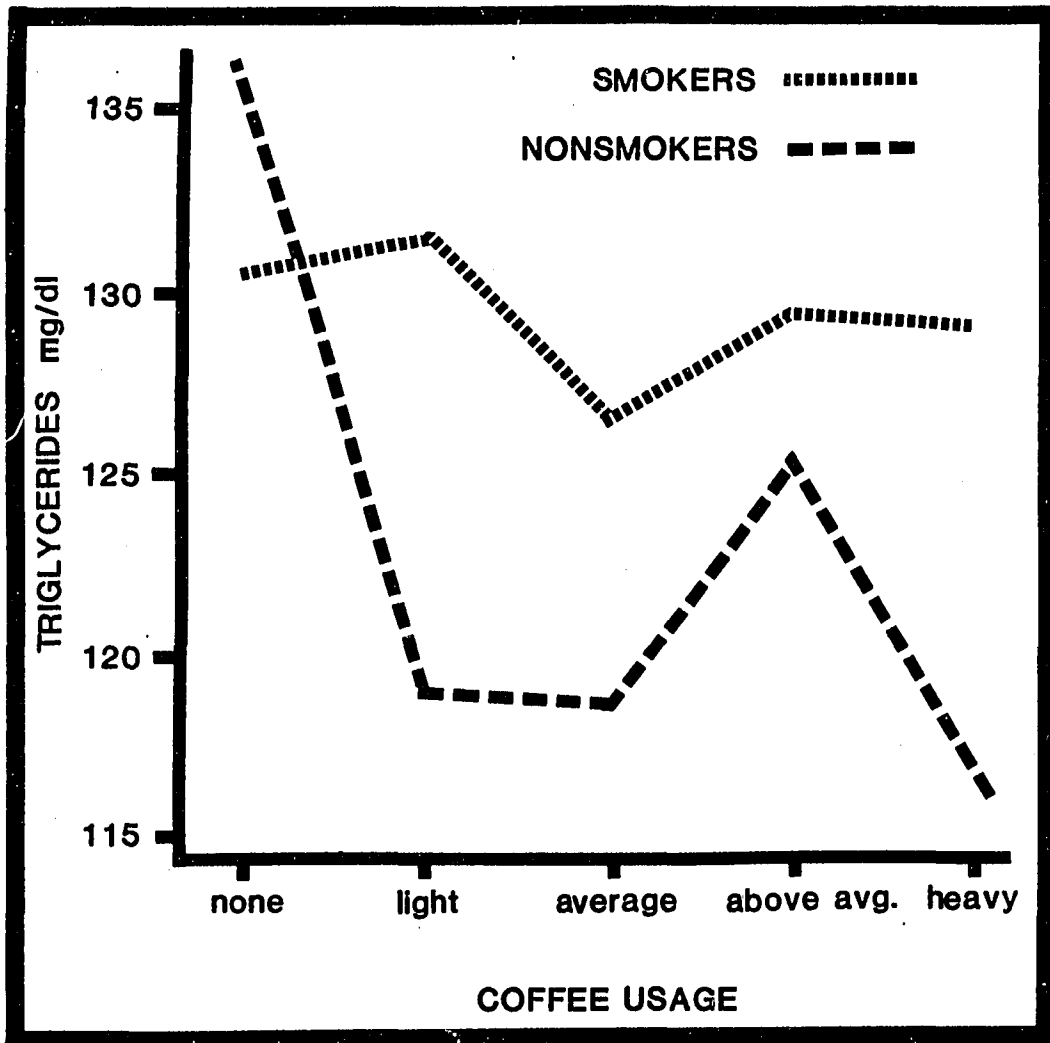


Figure 2. Triglyceride means by coffee usage and smoking status

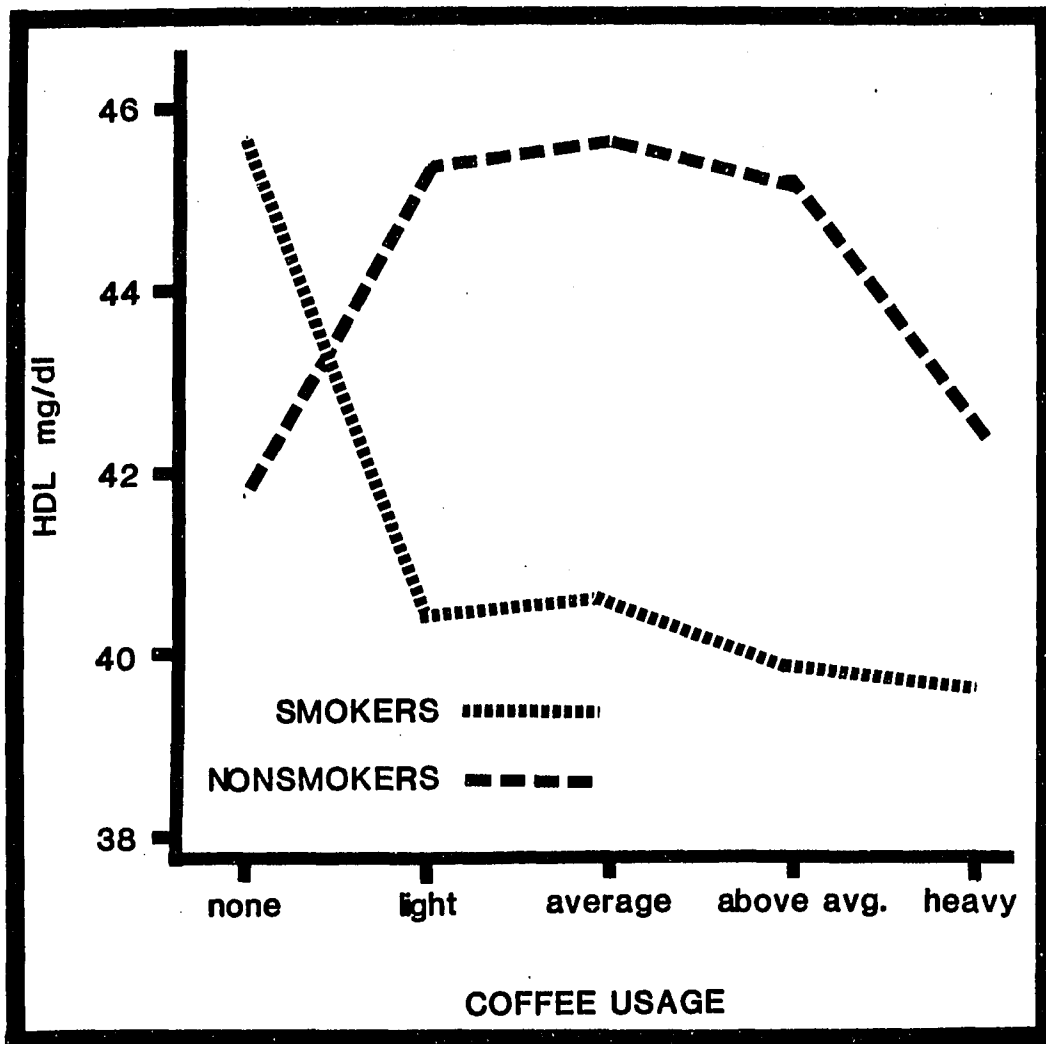


Figure 3. HDL means by coffee usage and smoking status

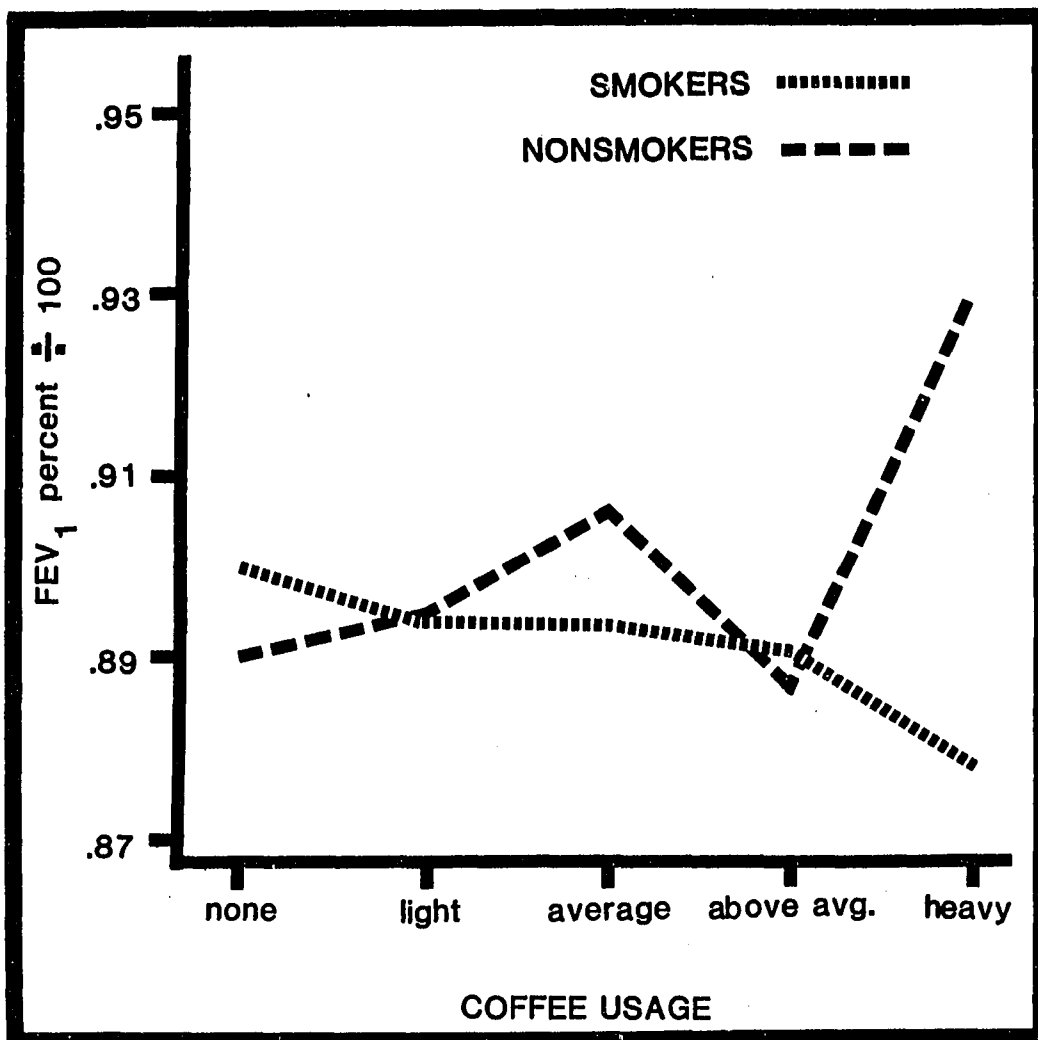


Figure 4. FEV₁ means by coffee usage and smoking status

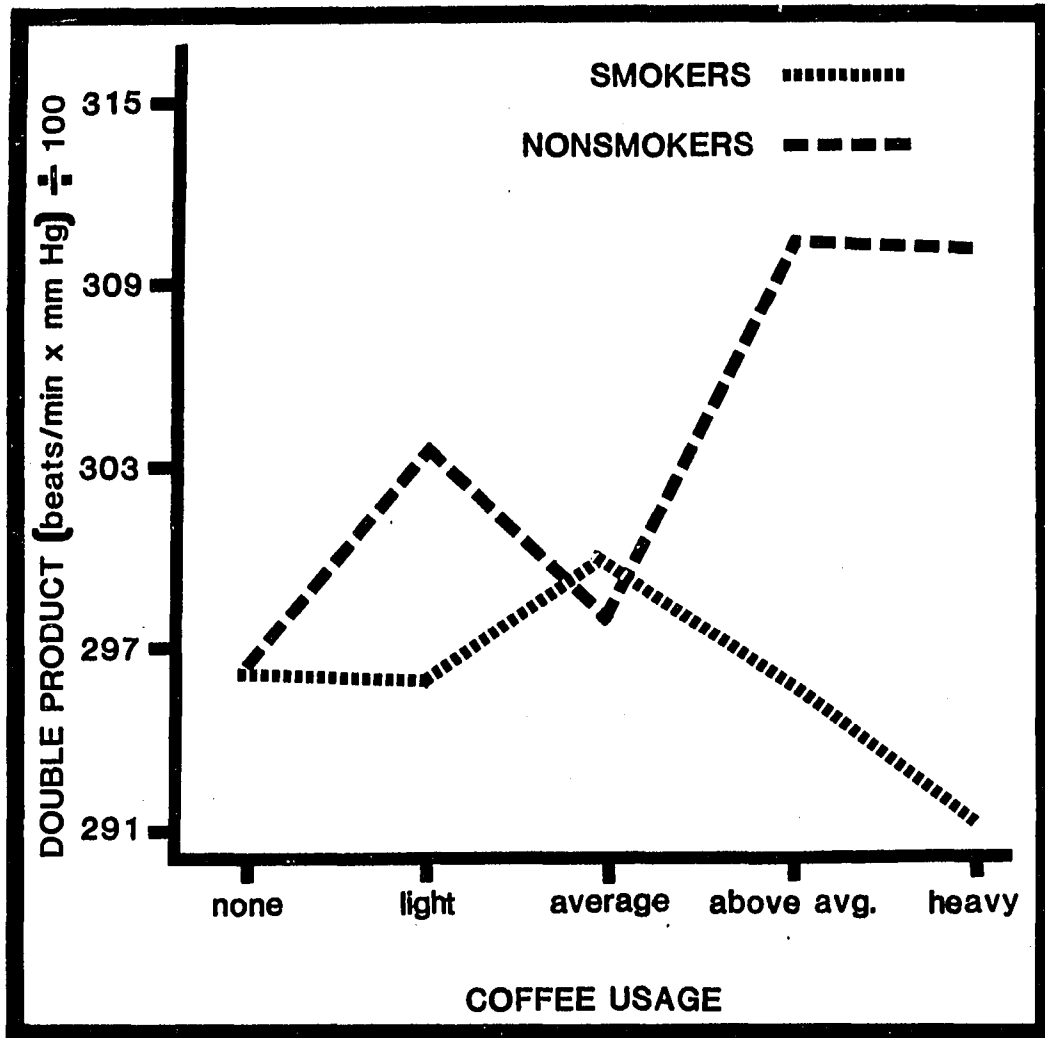


Figure 5. Double product means by coffee usage and smoking status

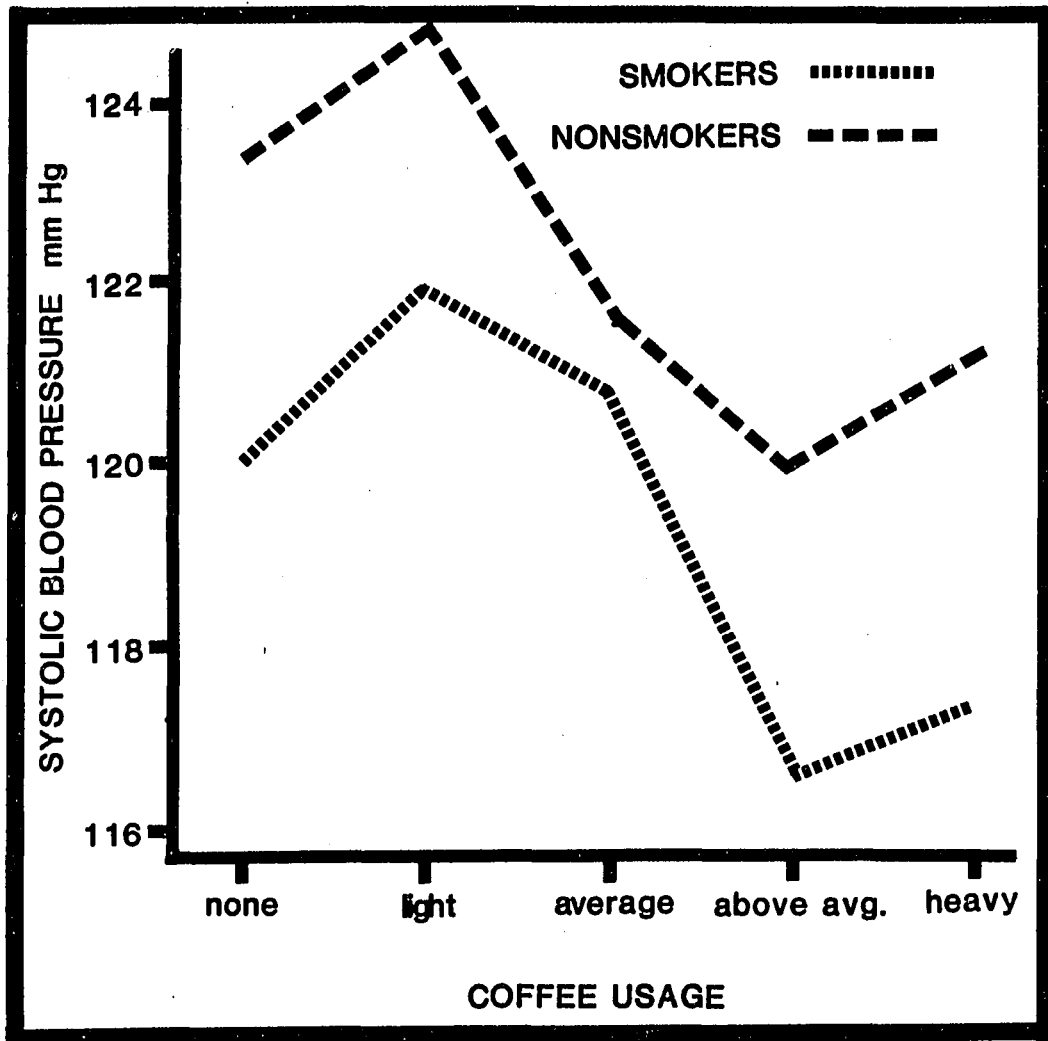


Figure 6. Systolic blood pressure means by coffee usage and smoking status

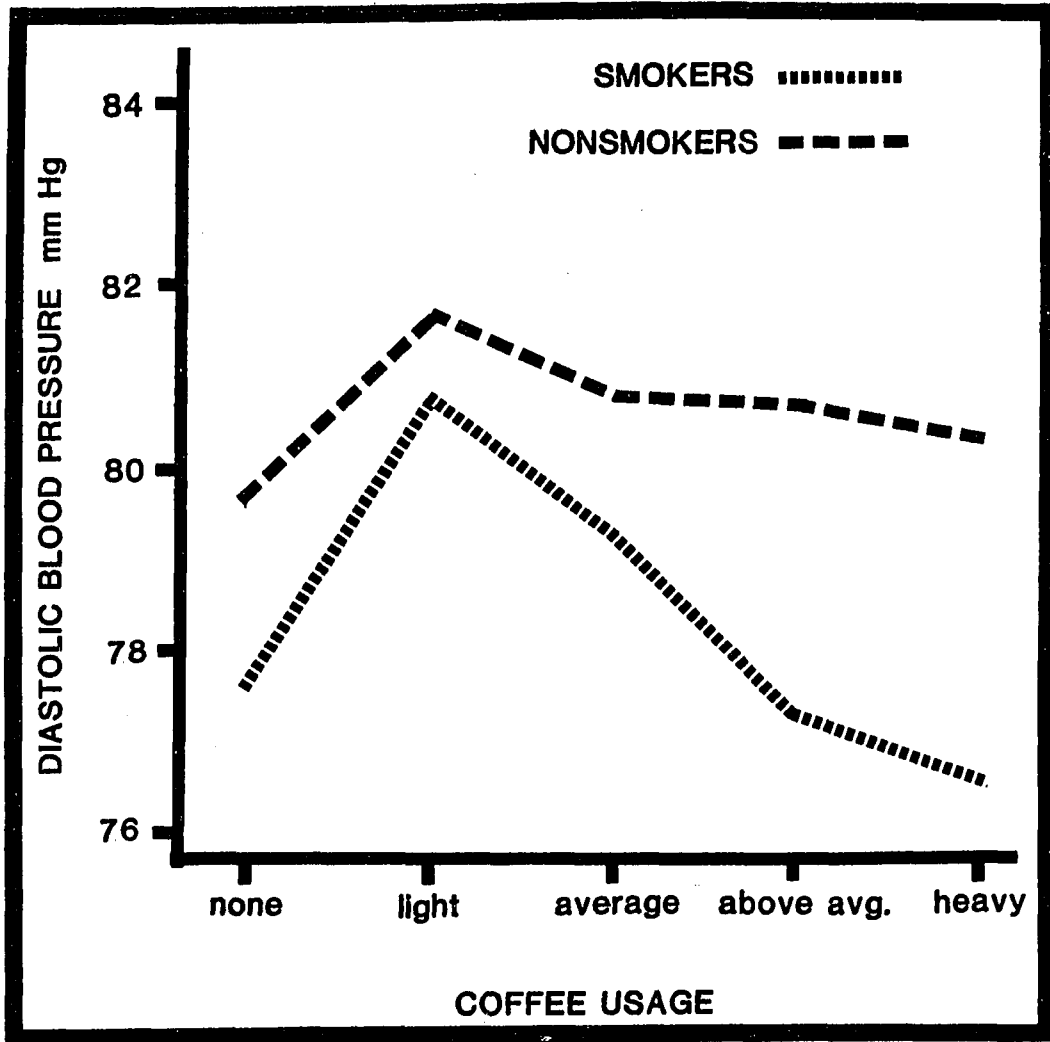


Figure 7. Diastolic blood pressure means by coffee usage and smoking status

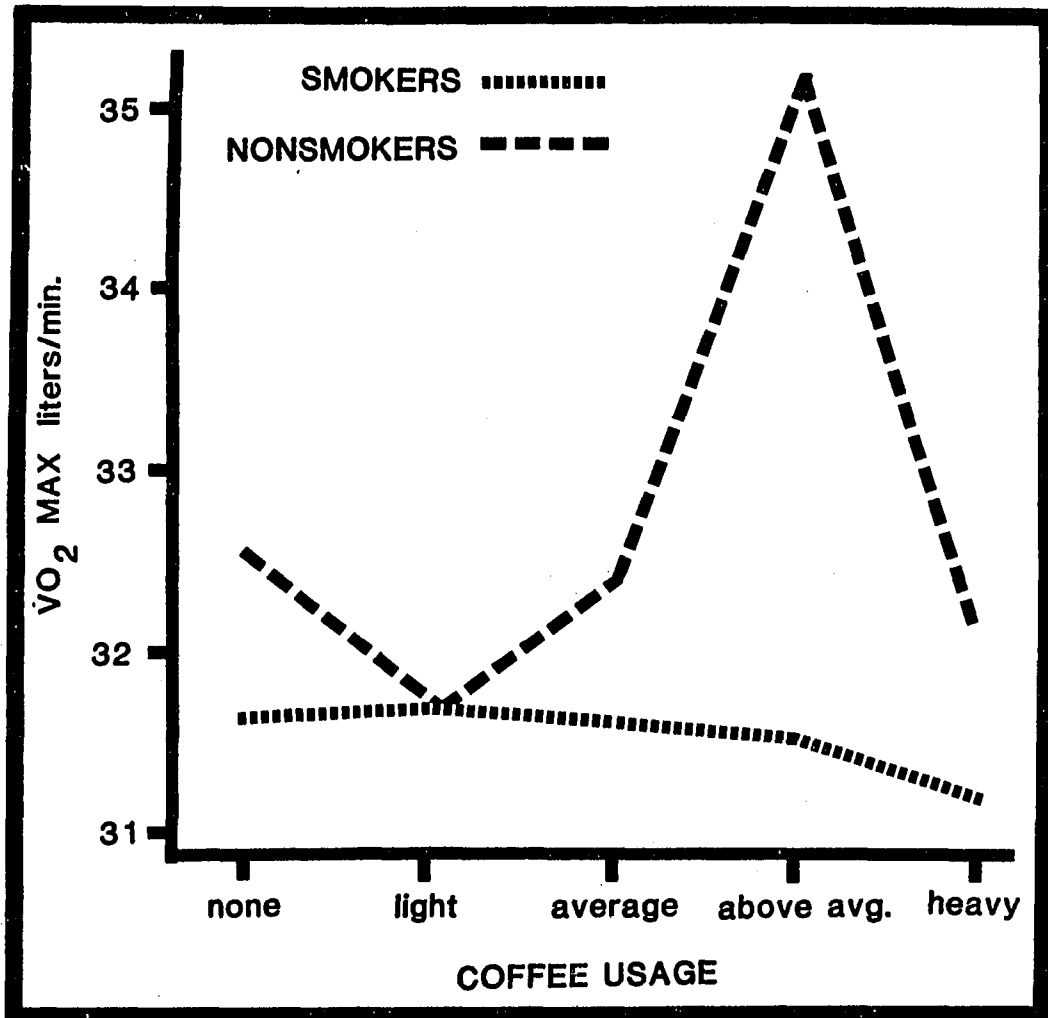


Figure 8, $\dot{V}O_2$ Max means by coffee usage and smoking status

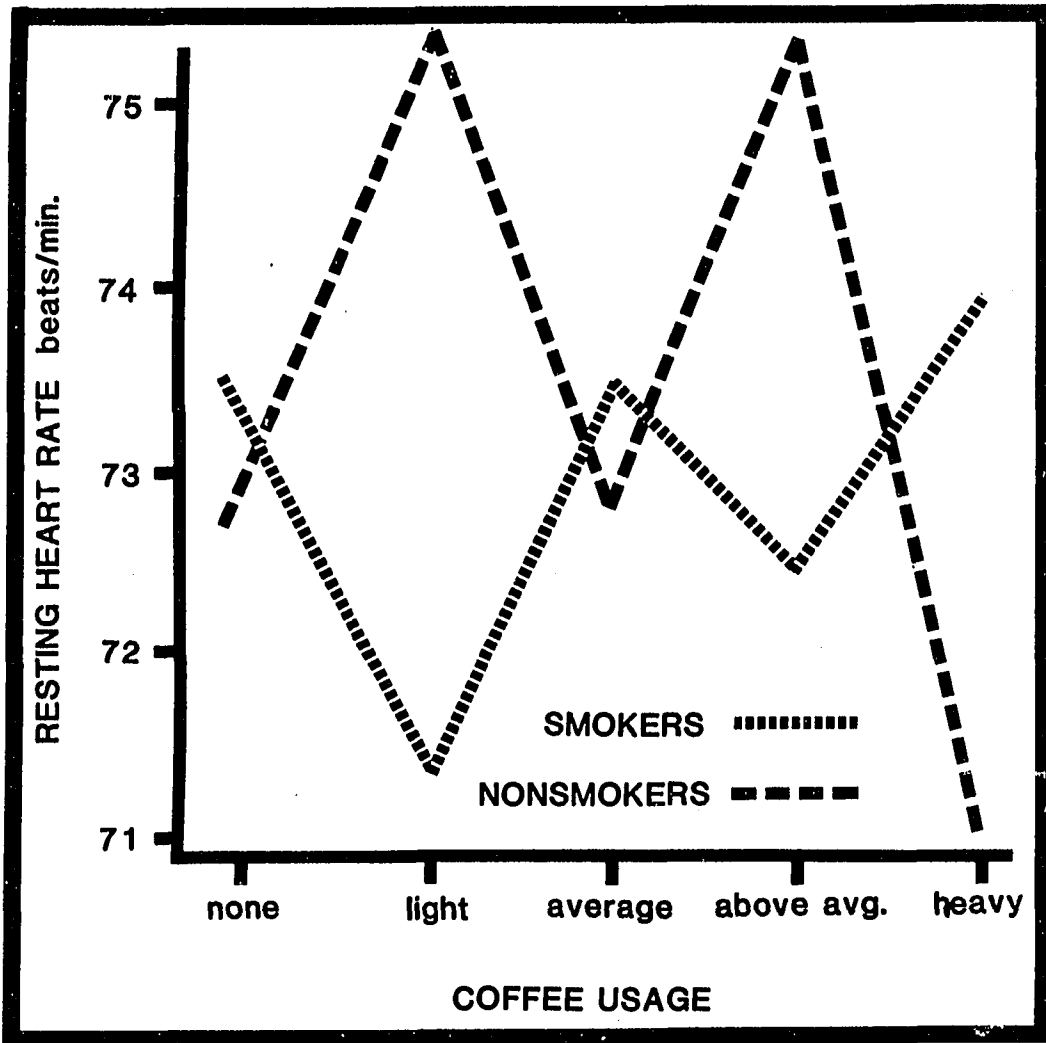


Figure 9. Resting heart rate means by coffee usage and smoking status

results of the univariate follow-up tests are presented in Table 5 for all nine dependent variables. Table 6 presents the main effect means and standard errors for coffee usage. The diagonal elements of the hypothesis and error matrices for the three independent effects are presented in Appendix D.

The results of the stepwise discriminant function demonstrated virtually no redundancy among the four variables found to differ for smokers and nonsmokers except in the case of systolic and diastolic blood pressure. Since the relationship between systolic and diastolic blood pressure is well documented, it was decided that once one of the blood pressures was selected for entry into the model, the other would be entered automatically. The procedure entered all four variables with the order of entry being HDL, diastolic and systolic blood pressure, and $\dot{V}O_2$ max. This was expected since the three physiological parameters of blood lipids, hemodynamics, and respiratory efficiency are known to be independent.

Smoking status explained 2.6 percent of the variance in HDL above that variance accounted for by all other variables in the model. The amount of variance explained by smoking status for $\dot{V}O_2$ max, diastolic, and systolic blood pressure was 0.9, 1.5, and 0.9 percent, respectively.

Follow-up analysis

Normal probability plots of residuals and scatter plots of residuals and predicted values indicated normality of errors and equality of variance for all nine dependent variables. Since the analysis of all possible interactions between covariates, smoking status,

Table 5
Univariate Analyses of Covariance
for Smoking Status

Dependent variable	Adjusted mean (Standard error)		<u>F</u>	<u>p</u>
	Smokers	Nonsmokers		
Chol	205.64 (2.56)	210.42 (2.34)	1.87	.1721
Trig	129.98 (4.62)	123.03 (3.96)	1.29	.2561
HDL	40.03 (.765)	44.01 (.761)	13.26	.0003
FEV ₁	.89 (.005)	.90 (.004)	2.49	.1154
DbProd	295.34 (3.10)	302.20 (2.80)	2.65	.1041
SysBP	119.39 (.806)	121.91 (.745)	5.16	.0236
DiaBP	78.11 (.544)	80.37 (.506)	9.08	.0027
$\dot{V}O_2$ max	31.36 (.356)	32.69 (.322)	7.51	.0064
RstHR	72.92 (.812)	73.89 (.744)	0.76	.3827

Note. df = (1,468) for all tests

Table 6
Adjusted Main Effect Means and Standard Errors
for Coffee Usage

Dependent variable	Coffee usage				
	None	Light	Average	Above avg.	Heavy
Chol	205.06 (4.44)	208.96 (3.66)	208.10 (3.14)	210.99 (3.52)	207.04 (4.60)
Trig	136.47 (8.44)	123.71 (6.26)	122.03 (5.31)	127.15 (6.15)	123.47 (7.88)
HDL	41.90 (1.39)	42.88 (1.17)	42.82 (1.00)	41.72 (1.08)	40.59 (1.39)
FEV ₁	.89 (.008)	.89 (.007)	.90 (.006)	.89 (.007)	.90 (.008)
DbProd	294.79 (5.38)	299.71 (5.40)	298.48 (3.79)	302.00 (4.21)	298.58 (5.55)
SysBP	121.87 (1.43)	123.20 (1.18)	121.10 (1.00)	118.20 (1.09)	118.93 (1.44)
DiaBP	78.79 (.954)	80.82 (.801)	79.90 (.681)	78.78 (.746)	77.91 (.973)
VO ₂ max	32.01 (.620)	31.44 (.507)	31.93 (.436)	33.11 (.485)	31.62 (.639)
RstHR	72.87 (1.41)	74.35 (1.18)	73.05 (1.00)	73.68 (1.11)	73.07 (1.46)

and coffee usage would not be practical nor theoretically based, only selected interactions previously suspect by the literature were examined.

Univariate analysis revealed a statistically significant interaction between age and smoking status for FEV_1 . As suggested by previous research, the effects of smoking on FEV_1 are only evident after longer exposure to smoking. Airway abnormalities brought about by smoking occur at a later age as a result of long term exposure. No other two factor interactions were found.

Summary

All of the research hypotheses were supported by the results except in the case of $\dot{V}O_2$ max. The null hypothesis of no differences between smokers and nonsmokers was not tenable for this dependent variable. The magnitude of hypothesized differences between smokers and nonsmokers for HDL, systolic, and diastolic blood pressure was very close to actual differences found in the data. Smoking effects tend to be additive across various degrees of coffee usage. Differences in FEV_1 between smokers and nonsmokers tend to be greater as age increases. Residuals from the regression models were normally and independently distributed with constant variance.

DISCUSSION

Coffee consumption

In contrast with early reports of a possible link between coffee consumption and coronary heart disease (Boston Collaborative Drug Surveillance Program, 1972; Jick et al., 1973; Paul, Lepper, Phelan, Dupertuis, MacMillan, McKean & Park, 1963), recent prospective and retrospective investigations have not found an association between coffee and CHD (Dawber et al., 1974; Hennekens et al., 1976; Heyden et al., 1978; Klatsky, Friedman & Siegelau, 1974; Yano et al., 1977). The present multivariate analysis found that coffee drinking was not significantly related to select CHD risk factors after controlling for age, weight, percent body fat, activity level and cigarette usage. This finding is consistent with several other studies that have failed to show permanent detrimental health effects produced by drinking coffee (see MacCornack, 1977). Sufficient evidence has accumulated to suggest that coffee drinking is not a precursor to CHD and is not mediated through other health effects frequently attributed to the development of CHD. In agreement with editorials by Vaisrub (1975-a,b) and Regan (1979), the time has come to exonerate coffee as a CHD risk factor, at least when used in moderation and in the absence of other risk factors.

Cigarette smoking

Certain conditions and diseases have been shown to occur more frequently in smokers than nonsmokers. The association of prolonged cigarette smoking with lung cancer, chronic bronchitis, and emphysema

is well-established, with causal relationships being accepted by many. Other relationships such as that between smoking and CHD are less clear. In areas where there is a high prevalence of CHD, studies consistently report a higher CHD incidence in smokers than nonsmokers (Doyle, Dawber, Kannel, Heslin & Kahn, 1962; Gordon, Garcia-Palmieri, Kagan, Kannel & Shiffman, 1974; Hammond & Horn, 1958; Pooling Project Research Group, 1978; Reid, McCartney, Hamilton, Rose, Jarrett & Keen, 1976). In addition, some of these studies have found the effects of smoking and other risk factors such as hypercholesteremia and arterial hypertension to be interactive rather than additive, in a synergistic manner (Gordon et al., 1974; Keys, 1970; Pooling Project Research Group, 1978; Reid et al., 1976). Conversely, countries with low rates of CHD and concomitantly low concentrations of serum cholesterol and atherosclerosis have shown little effect of heavy smoking on CHD incidence (Gordon et al., 1974; Keys, 1970). Although by no means substantiated, speculation as to the causal mechanisms involved generally center on the effects of nicotine and carbon monoxide on hemodynamics, blood lipids, and the atherosclerotic process (see Ball & Turner, 1974; Doyle, 1979). If, indeed, smoking does affect the cardiovascular system, lipids, and metabolism, one would expect to see differences between smokers and nonsmokers on other CHD risk factors involving these physiologic and metabolic processes. Differences between smokers and nonsmokers on other CHD risk factors would add additional support for the view that the influence of smoking on CHD was mediated through these risk factors.

The present study found statistically significant differences in $\dot{V}O_2$ max, HDL-c, systolic, and diastolic blood pressure between smokers and nonsmokers. No differences were found in FEV₁, resting heart rate, serum cholesterol, triglycerides, or double product.

The finding of a significant increase in $\dot{V}O_2$ max for nonsmokers, although small, is surprising since the majority of the studies which have evaluated this variable across smokers and nonsmokers have not uncovered a difference (Chevalier et al., 1963; Frayser, 1974; Krumholz et al., 1964; Krumholz et al., 1965). This study differed from other studies, however, in that the possible confounding effects of concomitant variables were statistically controlled. A more important difference is in the size of the samples. Relatively small samples of smokers and nonsmokers were evaluated in the majority of the studies assessing response to exercise. These small samples are probably a result of the time, effort, and personnel required to obtain a valid graded exercise test. Similar to the results obtained when evaluating FEV₁, differences in $\dot{V}O_2$ max are probably directly related to length and intensity of exposure. If cigarette smoking does impair ventilatory function, then it is possible that development of CHD could be mediated through respiratory efficiency. Further studies using larger sample populations are needed, especially in the case of $\dot{V}O_2$ max, before valid conclusions are possible. Measurement error plagues both $\dot{V}O_2$ max and FEV₁. Since both tests are based on maximal efforts, subjects who complete the treadmill schedule prematurely, or do not achieve maximal expiration, bias the results (Cumming, 1968).

It is curious that the double product and resting heart rate did not significantly differ between smokers and nonsmokers given that $\dot{V}O_2$ max was higher in nonsmokers. Interpretation of these findings for double product is reserved, since some subjects might not have attained their maximum heart rate. In addition, the difficulty of measuring systolic blood pressure while a subject is running on the exercise treadmill has previously been noted (Henschel, Vega & Taylor, 1954). Apparently even though $\dot{V}O_2$ max and cardiac rate are both measures of cardiac fitness, they are not always reflective of one another, at least with regards to smoking status.

The finding that systolic and diastolic blood pressures were lower among smokers than nonsmokers has been repeatedly found (Goldbourn & Medalie, 1977; Gyntelberg & Meyer, 1974; Seltzer, 1980). Why this relationship seems to exist is unknown. Researchers who observe this effect provide little insight as to the causal mechanism. The data presented here suggest that differences in blood pressures are in a direction likely to be beneficial to smokers. This relationship tends to negate the hypothesis that the effects of cigarette smoking on the incidence of CHD is mediated through blood pressure.

Although the present study found cholesterol and triglyceride levels to be similar for smokers and nonsmokers, HDL-c values were significantly lower in smokers. Studies which examine cholesterol and triglyceride levels are hindered by the fact that the variance in the laboratory methods is twice that of the difference usually being examined (see Appendix C). It is not unusual for triglyceride

levels to range from 20 mg/dl to 1000 mg/dl and up. Detecting consistent differences in HDL-c levels between smokers and nonsmokers is easier due to the higher precision in the laboratory determinations. The higher HDL levels in nonsmokers of 4 mg/dl is consistent with a range of about 2 to 6 mg/dl found by other researchers (Garrison et al., 1978; Heyden et al., 1979; Morrison et al., 1979), and is noteworthy when considering that the effects of body composition and activity level were removed. It has been suggested that smoking might affect CHD by unfavorably changing the relative proportion of the cholesterol components among smokers. Although several observational studies have observed an inverse relationship between HDL-c and smoking, causal relationships cannot be inferred. In fact, a recent attempt to establish a causal pathway was unable to show that the effect of smoking on HDL-c actually contributed to subsequent CHD (Criqui, Cowan, Tyroler, Bangdiwala, Heiss, Wallace & Davis, 1982).

Although rarely addressed, the issue of statistical significance versus clinical significance is relevant to the differences sometimes found to exist between smokers and nonsmokers. When large-scale studies report statistically significant differences between smoking groups on specific indices of health, these differences are almost always extremely small. In this study, although significant, the adjusted mean HDL-c values differed between smokers and nonsmokers by only 4 mg/dl, systolic and diastolic blood pressure by 2-3 mg Hg, and $\dot{V}O_2$ max by 1 liter/min. In addition, the amount of variance explained by whether a person smoked or not was less than 3 percent for HDL-c

and systolic blood pressure and less than 1 percent for diastolic blood pressure and $\dot{V}O_2$ max. Although these differences are real, they are unlikely to be important from a biological and clinical standpoint. Even when several variables that significantly discriminate between smokers and nonsmokers were combined, Goldbourt and Medalie (1977) reported the discriminating power to be only slightly better than random classification. The authors rightfully conclude that "the large assembly of 'significant t-values' in the literature may have masked what really constitutes very little discrimination" (Goldbourt & Medalie, 1977, p. 84). A similar finding has been reported by Thomas, Ross and Higenbothom (1964) among healthy medical students.

A review of the literature reveals almost as many studies not finding differences between smokers and nonsmokers on specific health indices and measurements as those that do. As has been noted, differences that are found are small. If smoking does, indeed, effect morbidity and mortality, it would appear either that its effects are mediated through health factors not yet identified or that there is a need for more precise measurements, as in the case of ventilatory function.

Conclusions

Within the limitations of any observational study (Cochran, 1972), no evidence was found for an effect of coffee on various health factors often associated with chronic diseases. The finding of no interaction between coffee drinking and smoking tends to support the premise

that coffee drinking is not harmful. Although the present data suggest that smokers are less physically fit, have lower blood pressures, and lower levels of HDL-c, these differences are extremely small, and are not likely to be important mediating factors in the developing of future morbidity and mortality. Given that these differences are probably real, the necessary evidence needed to establish a causal connection to CHD has yet to be established (Blackburn, Brozek, Taylor & Keys, 1960; Seltzer, 1980).

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The Iowa State University Committee on the Use of Human Subjects in Research reviewed this project and concluded that the rights and welfare of the human subjects were adequately protected, that risks were outweighed by the potential benefits and expected value of the knowledge sought, that confidentiality of data was assured and that informed consent was obtained by appropriate procedures.

APPENDIX A:
INFORMED CONSENT FORM
Iowa State University
Exercise Clinic
Ames, Iowa

All information is private and confidential, except for statistics.

Name _____
 First Middle Last

Address _____
 Street City State Zip

Phone _____ SS# _____ Date _____

Family Physician _____

Physician's Address _____ Phone _____

APPENDIX B:
GENERAL INFORMATION QUESTIONNAIRE

Marital Status

Single Married Divorced Widowed Separated

Age _____ Sex _____

What is your level of education?

Grade School Jr. High School High School
 Some College College Graduate Graduate School
 Vocational Other _____ (specify) _____

If married, what is your spouse's level of education?

Grade School Jr. High School High School
 Some College College Graduate Graduate School
 Vocational Other _____ (specify) _____

Racial or Ethnic background?

White Black (Afro-American) American Indian
 Asian or Pacific Islander Chicano (Mexican American)
 Other _____

Please describe the type of work you do.

OCCUPATION (salesperson, physician, loan officer, etc.)

TYPE OF ORGANIZATION (department store, private clinic, commercial bank)

If married, please describe the type of work your spouse does.

OCCUPATION

TYPE OF ORGANIZATION

In general, what degree of mental/emotional stress do you experience on the job?

No Stress Extreme Stress
 1 2 3 4 5 6

In general, what degree of physical stress do you experience on the job?

No Stress Extreme Stress
 1 2 3 4 5 6

Would you classify yourself as a nervous person?

Not Nervous Very Nervous
 1 2 3 4 5 6

Does your job give you a chance to use your abilities and talents?

59

Very Much; Quite a bit; Some; Little; None

Do you get a feeling of accomplishment from the work you are doing?

Very much; Quite a bit; Some; Little; None

If I were to describe myself, you would get a better understanding of me through my leisure time activities than through my work?

Very strongly agree; Moderately agree; Slightly agree; Slightly disagree;

Moderately disagree; Very strongly disagree

My personal identity is realized more in my work than in my leisure time?

Very strongly agree; Moderately agree; Slightly agree; Slightly disagree;

Moderately disagree; Very strongly disagree

In general, how satisfying do you find the way you are spending your life these days?

Very Satisfying; Moderately Satisfying; Slightly Satisfying;

Slightly Dissatisfying; Moderately Dissatisfying; Very Dissatisfying

Taking all things together, how would you say things are going these days?
Would you say that you are:

Very Happy; Moderately Happy; Slightly Happy; Slightly Unhappy;

Moderately Unhappy; Very Unhappy

Reason for Performing Stress Test

- | | |
|---|--|
| <input type="checkbox"/> Chest Pain | <input type="checkbox"/> Arrhythmia Eval. |
| <input type="checkbox"/> Screening for IHD | <input type="checkbox"/> Functional Capacity Eval. |
| <input type="checkbox"/> Angina Pectoris Eval. | <input type="checkbox"/> Iowa Highway Patrol Program |
| <input type="checkbox"/> Post M.I. Rehabilitation | |

Check the box in front of those questions to which your answer is yes. Leave others blank.

- Has a doctor ever said that your blood pressure was too high or too low?
 Do you ever have pain in your heart or chest?
 Are you often bothered by a thumping of the heart?
 Does your heart often race like mad?
 Do you ever notice extra heart beats or skipped beats?

- Are your ankles often badly swollen?
- Do cold hands or feet trouble you ever in hot weather?
- Has a doctor ever said that you had or have heart trouble, an abnormal electrocardiogram (ECG), heart attack or coronary?
- Do you suffer from frequent leg cramps?
- Do you often have difficulty breathing?
- Do you get out of breath long before anyone else?
- Do you sometimes get out of breath when sitting or sleeping?
- Has a doctor ever told you your cholesterol level was high?
- Has a doctor ever told you your triglyceride level was high?

Comments: _____

Date of last Physical Examination:

_____ _____ Never Normal Abnormal

Month Year

Date of last chest x-ray:

_____ _____ Never Normal Abnormal

Month Year

Date of last electrocardiogram:

_____ _____ Never Normal Abnormal

Month Year

Have you ever had:

- | | |
|---|---|
| <input type="checkbox"/> Heart Attack, how many ago? _____ | <input type="checkbox"/> Recent embolism |
| <input type="checkbox"/> Rheumatic Fever | <input type="checkbox"/> Asthma |
| <input type="checkbox"/> Heart Murmur | <input type="checkbox"/> Thyroid Problems |
| <input type="checkbox"/> Diseases of the arteries | <input type="checkbox"/> Pneumonia |
| <input type="checkbox"/> Varicose Veins | <input type="checkbox"/> Bronchitis |
| <input type="checkbox"/> Arthritis | <input type="checkbox"/> Other Lung Diseases |
| <input type="checkbox"/> Thrombo | <input type="checkbox"/> Injuries to Back, Arms, legs or joints |
| <input type="checkbox"/> Phlebitis | <input type="checkbox"/> Broken Bones |
| <input type="checkbox"/> Dizziness or Fainting Spells | <input type="checkbox"/> Jaundice or Gallbladder problems |
| <input type="checkbox"/> Epilepsy or Fits | <input type="checkbox"/> Polio |
| <input type="checkbox"/> Strokes | <input type="checkbox"/> Urinary Tract Infections |
| <input type="checkbox"/> Scarlet Fever | <input type="checkbox"/> Nervous or Emotional Problems |
| <input type="checkbox"/> Anemia | |
| <input type="checkbox"/> Increasing angina pectoris with effort | |

Do you now have or have you recently had:

- A chronic, recurrent or morning cough?
- Any episode of coughing up blood?
- Increased anxiety or depression?
- Problems with recurrent fatigue?
- Problems sleeping or increased irritability?
- Migraine or recurrent headaches?
- Swollen or painful knees or ankles?
- Swollen, stiff or painful joints?
- Pain in your legs after walking short distances?
- Back pain?
- Kidney problems such as passing stones, burning, increased frequency, decreased force of stream or difficulty in starting or stopping your stream?
- A cold or other infection?
- A persistent illness?
- Prostate trouble (men only)?
- Any stomach or intestinal problems such as recurrent heartburn, ulcers, constipation or diarrhea?

Comments: _____

Are you presently taking any of the following medication?

- | | |
|---|--------------------------------------|
| <input type="checkbox"/> Antihypertensive | <input type="checkbox"/> Propranolol |
| <input type="checkbox"/> Nitrates | <input type="checkbox"/> Quinidine |
| <input type="checkbox"/> Digitallis | <input type="checkbox"/> Thyroid |
| <input type="checkbox"/> Pronestyl | <input type="checkbox"/> |
| <input type="checkbox"/> Dilantin | <input type="checkbox"/> |
| <input type="checkbox"/> Diuretics | <input type="checkbox"/> None |
| <input type="checkbox"/> Drug for Allergies _____ | |

List any other prescribed medication you are now taking: _____

List any self-prescribed medications or dietary supplements you are now taking regularly: _____

Comments: _____

Family Medical History

Father: Alive Current Age _____ Deceased Age of death _____
General health now: Excellent Good Fair Poor Don't know
Cause of death _____

Mother: Alive Current age _____ Deceased Age of death _____
General health now: Excellent Good Fair Poor Don't know
Cause of death _____

Have any of your blood relatives had any of the following? (Include brothers, sisters, grandparents, uncles and aunts)

- Heart Attacks, under age 50
- Strokes, under age 50
- High blood pressure
- Elevated Cholesterol
- Diabetes
- Asthma or Hay Fever
- Congenital heart disease
- Heart Operations
- Obesity (20 or more lbs. over-weight)

Smoking

Have you ever smoked cigarettes, cigars or a pipe? Yes No

Do you presently smoke? Yes No

Age you started smoking: _____

If you did or do smoke cigarettes, how many per day? _____

If you did or do smoke cigars, how many per day? _____

If you did or do smoke a pipe, how many per day? _____

If you have quit smoking, when was it? _____ year

Diet

What do you consider your ideal weight? _____ lbs.

What is the most you ever weighted? _____ lbs. Age _____

Weight: Now _____ lbs. One year ago _____ lbs. At age 18 _____ lbs.

Average number of eggs you usually eat per week _____

Number of times per week you usually eat:

Beef _____	Fish _____	Desserts _____
Pork _____	Fowl _____	French Fried Foods _____

Salt Consumption: Heavy Moderate Light None

Number of servings (cups or glasses) per week you usually consume:

Whole Milk _____	Buttermilk _____	2% Milk _____
Skim Milk _____	Tea (hot or iced) _____	Coffee _____

Do you ever drink alcoholic beverages? Yes No

If yes, what is your approximate intake of these beverages?

	None	Occasional	Often
Beer	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Wine	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Hard Liquor	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

At any time in the past were you a heavy drinker (consumption of 6 oz. of hard liquor per day or more)? Yes No

Comments _____

Following are listed a number of things characteristic of yourself or related to you. Consider each item listed and write in the number which best represents your feelings according to the following scale:

1. Have strong positive feelings
2. Have moderate positive feelings
3. Have no feeling one way or the other.
4. Have moderate negative feelings.
5. Have strong negative feelings.

1. appetite	_____	13. width of shoulders	_____
2. physical stamina	_____	14. arms	_____
3. muscular strength	_____	15. chest (or breasts)	_____
4. waist	_____	16. digestion	_____
5. energy level	_____	17. hips	_____
6. back	_____	18. resistance to illness	_____
7. age	_____	19. legs	_____
8. body build	_____	20. sleep	_____
9. profile	_____	21. health	_____
10. height	_____	22. posture	_____
11. keenness of senses	_____	23. face	_____
12. tolerance for pain	_____	24. weight	_____

Exercise and Sports Information

Are you currently involved in a regular program of physical activity?

Yes No

If yes, how long have you been involved in your program? _____

Give three reasons why you became involved in your program of physical activity.

1. _____
- _____
- _____
2. _____
- _____
- _____

3. _____

What kind of successes or failures have you had? _____

Have you experienced any injury problems related to your physical activity?
 Yes No

Do you regularly (at least 3 times per week) walk or run one or more miles continuously? Yes No

If yes, average number of miles you cover per workout or day. _____ miles

What is your average time per mile? _____ (min:sec)

Do you practice weight lifting or home calisthenics? Yes No

Are you involved in an aerobic points per week exercise program Yes No

If yes, what is your average points per week? _____

Have you participated in either of the following in the past six months?

12 Minute Test _____ Number of miles in 12 minutes

1.5 Miles _____ Min:Sec Time for 1.5 miles

Did you participate in high school competitive sports? Yes No

If yes, which one or ones?

- | | | | |
|--|-------------------------------------|-------------------------------------|------------------------------------|
| <input type="checkbox"/> Track | <input type="checkbox"/> Swimming | <input type="checkbox"/> Tennis | <input type="checkbox"/> Wrestling |
| <input type="checkbox"/> Cross Country | <input type="checkbox"/> Golf | <input type="checkbox"/> Gymnastics | |
| <input type="checkbox"/> Football | <input type="checkbox"/> Basketball | <input type="checkbox"/> Baseball | |
| <input type="checkbox"/> Other _____ | | | |

Did you participate in college competitive sports? Yes No

If yes, which one or ones?

- | | | | |
|--|-------------------------------------|-------------------------------------|------------------------------------|
| <input type="checkbox"/> Track | <input type="checkbox"/> Swimming | <input type="checkbox"/> Tennis | <input type="checkbox"/> Wrestling |
| <input type="checkbox"/> Cross Country | <input type="checkbox"/> Golf | <input type="checkbox"/> Gymnastics | |
| <input type="checkbox"/> Football | <input type="checkbox"/> Basketball | <input type="checkbox"/> Baseball | |
| <input type="checkbox"/> Other _____ | | | |

Within the past year, list the sport or physical activity/activities which you have participated in. Also list frequency of participation in each activity.

	Activity	Frequency/week
1.	_____	_____
2.	_____	_____
3.	_____	_____
4.	_____	_____
5.	_____	_____
6.	_____	_____

With whom do you participate (spouse, fellow worker, neighbor, etc.)?

Following are listed a number of reasons for your present or continued participation in the Exercise Clinic. Consider each item listed and write in the number to the response that best represents your feeling according to the listed scale:

- | | |
|------------------------|-----------------------|
| 1. Extremely important | 3. Of some importance |
| 2. Important | 4. Of no importance |

- ____ It is more stimulating than my job
- ____ It helps relieve my tensions
- ____ It provides an escape from the pressure of work
- ____ It gives me a feeling of freedom
- ____ It allows me to be with people I like
- ____ It's a chance to do something different
- ____ It helps me to keep physically fit
- ____ It gives me a chance to meet new people
- ____ I can demonstrate my achievements to others
- ____ It's nice to tell others about it
- ____ It's a chance to be alone
- ____ It's organized or semistructured
- ____ Encouraged to work hard
- ____ Reaching a specific goal
- ____ Lets me know when I do well
- ____ Shows us how to do things right
- ____ Other (Specify) _____
-
-

APPENDIX C:
CLINICAL LABORATORIES LETTER

CLINICAL LABORATORIES, P.C.
 11th and Duff
 P.O. Box 1683 - Ames, Iowa 50010
 (515)233-2316

*Practice limited to
 Laboratory Medicine
 and Pathology*

To: Linda Frankenberger, Director
 Physical/Occupational Therapy

From: Jerry Sloan
 Chief Medical Technologist

Subject: Coefficient of variance for serum lipids

Date: 3/5/81

In accordance with the Joint Commission for Accreditation of Hospitals the Laboratory must meet very strict standards with regards to quality control i.e., precision, accuracy and consistency of laboratory tests. Replicate control standards for the SMAC are run every fifteen samples to correct for drift and to re-standardize the instrument. HDL manual chemistry determinations are completed in duplicate including the control standards and values are not reported unless the samples are in agreement within 95%. On a monthly basis, laboratory data is submitted to DADE, a division of Scientific Products, Miami, Florida, who prepares a computerized quality control summary. Within this report coefficients of variance of all laboratory tests are documented. The coefficients of variance and standard deviation for the serum lipids based on the January, 1981, report are listed below.

Total serum cholesterol	7.3	±	9.9
Total serum tryglycerides	7.8	±	5.2
High density lipoprotein cholesterol	1.37	±	1.21

APPENDIX D:

MULTIVARIATE STATISTICS

Diagonal Elements of the Hypothesis and Error Matrices

Variable	Coffee usage	Smoking status	Interaction
Hypothesis			
SRChol	1.932	2.914	3.599
LgTrig	.6135	.3203	.2103
LgHDL	.1598	.9498	.5325
LgtFEV ₁	1.018	1.288	5.460
DbProd	2382.	4983.	7674.
LgSysBP	.1005	.0462	.0099
LgDiaBP	.0683	.0864	.0179
VO ₂ max	167.9	187.2	176.1
LgRstHR	.0260	.0186	.1347
Error			
SRChol	730.0	730.0	725.4
LgTrig	115.9	115.9	115.7
LgHDL	33.53	33.53	33.00
LgtFEV ₁	242.4	242.4	236.9
DbProd	879550.	879550.	871876.
LgSysBP	4.187	4.187	4.177
LgDiaBP	4.452	4.452	4.434
VO ₂ max	11661.	11661.	11485.
LgRstHR	11.38	11.38	11.24